22.

Causes of Diseases and Death of Fishes in Captivity.

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(Plates I-VI).

Introduction.

This report deals with the several causes of mortality of fishes exhibited in The New York Aquarium at Battery Park, New York City. Attempts are made here to analyze the more important causes and to indicate, where possible, therapeutic and prophylactic measures. It is hoped that these records will be of some value in maintaining fish in good health and in preventing major losses.

The lists of causes of deaths for 1940 and for the first eight months in 1941 are shown in Tables I-III. It was soon after the latter date that the Aquarium closed its doors at the Battery and the fishes and other vertebrates and invertebrates were distributed to certain institutions or brought to the temporary Aquarium in the New York Zoological Park.

The agents causing death of specimens in the present temporary Aquarium are few and will not be discussed in detail. It is sufficient to report that the number of mortalities for 1942 was exceptionally low and the causes limited to such factors as handling, temperature and chemical changes and to such pathogens as *Saprolegnia*, *Mycobacterium* and *Ichthyophthirius*.

It may be of interest to point out that the terminology used in designating the causes of death of fishes follows, as much as possible, the "Manual of the International List of Causes of Death" for human beings published by the United States Department of Commerce (1939).

Analysis of the Immediate Causes of Death of Fishes.

I. General Remarks.

Table IV gives comparative figures on the number of monthly mortalities for the years 1939, 1940 and for the first eight months of 1941. The saturation of the population density suddenly with large collections of fishes explains, in part, the quick rise in the number of deaths at any given time. In 1939, a large shipment of marine fishes was received from Florida and placed on exhibition. The devastating effect of this procedure is reflected in the great number of losses shown for July and August of that year. No exceptional losses were incurred in 1940 and 1941, when only small shipments were received from time to time and the fishes were introduced gradually into the established population. Further, diseases that may have appeared were kept under control.

A similar picture can be demonstrated for the temperate fresh-water species. In 1939, collections of these forms were held down to a minimum, but when several big hauls were made in 1940, comprising mostly trout and other psychrophylic forms, parasitic diseases became rampant on the rise in temperature, and because of the crowded conditions, the high mortalities shown in June and July of that year were the result. When the susceptible fishes died off, the death rate once more approached the normal.

The steady death rate that is indicated for the tropical fresh-water fishes is accounted for by the fact that each tank is an independent system and diseases, therefore, are limited to the particular tank in which they appear. Furthermore, the population of these tanks is more carefully adjusted.

In general, there was a significant decrease (about 20%) in the total number of deaths among fishes in the Aquarium in 1940 as compared with the number of mortalities during 1939. Controlling the population density was the main factor in this decline.

Concomitant with the decrease in mortality was the increase in average longevity. The average life span in captivity was about 12 months in 1939, 15 months in 1940 and 17 months in 1941. A census taken in April, 1941, showed that 71% of the fishes on exhibition were under 2 years; 15% 2-5 years and 14% over 5 years.

II. Causes of Death of Marine Fishes.
(Table 1)

Among the marine species the decrease in mortalities was especially significant as shown in Table IV and, for the most part, is due to the almost complete disappearance

TABLE I.

CAUSES OF DEATH OF MARINE FISHES.

Parasitic and Infectious Diseases.

DISEASES	1940	1941
Diseases of Skin and Gills		
1. Bacterial	8	23
2. Oodinium (Flagellate)	11	—
3. Trichodina (Ciliate)	20	24
4. Myxosporidia (Cnidospordia)	—	3
5. Epibdella (Trematoda)	99	2
6. Microcotyle (Trematoda)	44	14
7. Diplectanus (Trematoda)	2	—
8. Argulus (Copepoda)	2	—
9. Livonica (Isopoda)	1	—
Diseases of Skin and Internal Organs		
10. Lymphocystis	9	—
Diseases of Digestive System		
11. Enteritis and Stenosis due to Acanthocephala	5	5
Diseases of Circulatory System		
12. Pericarditis due to Echinostome infection	—	1
Total	201	72

Non-Infectious and Non-Parasitic Diseases.

Neoplastic Diseases		
13. Nephroma	—	2
14. Thyroid Tumor	1	1
Diseases of Digestive System		
15. Prolapsed Intestine with Stenosis	1	—
16. Hepatic Degeneration	7	10
17. Fatty Degeneration of Liver	—	11
Diseases of Urinary System		
18. Kidney Degeneration	—	5
Diseases of Reproductive System		
19. Ovarian Degeneration	—	1
Diseases of Circulatory System		
20. Cardiac Degeneration	—	3
21. Fatty Degeneration of Heart	—	1
22. Ruptured Myocardium	1	—
23. Gas Embolism (Cerebral Hemorrhage)	61	4
24. Internal Hemorrhage	—	2
Diseases of Bone and Organs of Locomotion		
25. Tail atrophy	—	1
Diseases of Ill-defined Causes		
26. General Degeneration of Internal Organs	—	2
27. Edema	—	1
Violent and Accidental Deaths		
28. Killed in Fighting	26	22
29. Jumped Tank	2	3
30. Fractured Skull	1	2
31. Multiple Abrasions	10	8
Deaths Due to External Causes		
32. Temperature Changes	—	2
33. Changes in Water Chemistry	85	2
Diseases of Organ of Vision		
34. Blindness	9	1
Diseases Due to Nutrition		
35. Malnutrition	17	—
36. Fatty Degeneration	4	—
Senility		
37. Deaths Due to Old Age	—	4
38. Causes Unknown	67	35
Total Non-infectious Diseases	292	123
Total Infectious Diseases	201	72
Grand total	493	195

of *Oodinium ocellatum*, a parasitic dinoflagellate, as an important death producing agent. This organism, together with another protozoan form, *Trichodina*, and the monogenetic fluke, *Epibdella melleni*, was responsible in 1939 for more than 60% of the deaths. In 1940, these parasites were the cause of only 26% of the fatalities. Although there was a decrease in the number of deaths due to *Epibdella* (Fig. 2) in 1940, it was still a major cause, being responsible for 20% of the mortalities; up 2% over the previous year. The decrease in the number of deaths resulting from Epibdellaiasis was partly due to the fact that epidemics of this form were controlled by limiting and segregating highly susceptible fishes, particularly the members of the family Chaetodontidae, and by a consistent application of prophylactic and therapeutic measures. The continuance of these measures (limiting the collection of susceptible fishes, control of temperature and density of sea water) yielded effective results with almost complete disappearance in 1941 of this agent as an important cause of death.

In spite of an increase in the number of mortalities due to *Microcotyle*, this fluke was never a real threat because it failed at any time to reach epidemic proportions. The site of infestation is limited to the gills of a few species of chaetodontid fishes. Dipping infested fish in fresh-water of the same temperature as the sea water from which they were taken is a very effective treatment. In using this technique, care should be taken not to prolong the exposure to fresh-water beyond a certain time limit, which must be established for the various susceptible species.

It was stated by Ward (1908) that "the parasitic fauna is primarily a function of the habitat of the species" and that marine fishes (e. g., salmon) "carry their marine parasites with them into fresh water but soon lose them." It is this basic principle that is put into practical use above. In treating parasitized fresh-water fishes, sea-water is used in the same manner. This procedure is highly effective and with least danger to the host for those migratory species which move from fresh water to sea-water and back again (e. g., eel, trout, salmon, etc.).

Two species of *Trichodina* (*T. spheroides* Padnos and Nigrelli and *T. halli* P. and N.,

1942) were responsible for 10% of the deaths of marine fishes in 1939, 4% in 1940 and 12% in 1941. However, the 1941 deaths due to this peritrich ciliate were limited to puffers (*Sphaeroides maculatus*) which were segregated after it was determined that this host was the foci for deaths of other species. Nigrelli and Atz (1943) pointed out the importance of a routine check for parasites of fishes that are to become part of an established and healthy collection. The danger of introducing fish haphazardly was demonstrated strikingly when a routine examination revealed that the epidemic of trichodiniasis in 1939 had its origin in puffers taken from Sandy Hook Bay area. Oodiniasis also was centered in puffers and other fishes from this area (see Nigrelli, 1936). Knowledge of the principal host and the area from which the fisn are taken becomes an important item in controlling disease-producing organisms that may destroy other and more important exhibition specimens.

A. Other Causes of Death of Marine Fishes.

1. Bacterial

A large gram-positive bacillus was found associated with certain skin lesions present on sea robins, toadfish and striped bass. In the latter, the rods were recovered from the kidneys. Externally, the lesion appears as whitish patches on the dorsal part of the body. When the growth becomes extensive death results. These fish are ordinarily placed in filtered bay water (low sp. gr. and pH) and the disease appears in about ten days after they are brought in from local waters. Similar species kept in the closed circulation (high sp. gr. and pH) did not show this infection.

An infectious dermatitis of similar type, but caused by a gram-negative bacillus, was reported by Zobell and Wells (1934) for several marine species of the west coast.

2. Lymphocystis.

This disease, peculiar only to teleost fish, is of a much wider distribution than has been suspected. An intracellular virus is believed to be the causative agent and the lesions appear as nodular growths on the skin and fins of the host. Microscopically, the growth shows tremendously enlarged connective tissue cells. The real nature of this condition was first reported by Weissenberg in 1914 (see also Weissenberg, 1938) in certain European fishes. Lymphocystis was reported from marine fishes in the N. Y. Aquarium by Smith and Nigrelli (1937), Weissenberg, Nigrelli and Smith (1937), Nigrelli and Smith (1939) and by Nigrelli (1940). In these papers the following hosts were described as being affected: blue angelfish (*Angelichthys isabelita*), hogfish (*Lachnolaimus maximus*), orange filefish (*Ceratacanthus schoepfii*), and the clownfish (*Amphiprion percula*). The disease was not seen in 1941 but in 1940 nine cases were found in which the growth had invaded the internal organs, producing the identical lesions reported by Nigrelli and Smith (1939) for the orange filefish. The hosts involved in these cases are: orange filefish (*Ceratacanthus schoepfii*), banded butterflyfish (*Chaetodon striatus*), black angelfish (*Pomacanthus arcuatus*), and East Indian cowfish (*Ostracion cornutus*). In 1942, a cowfish (*Lactophrys tricornis*) sent to the writer by Dr. C. M. Breder from Palmetto Key, Florida, was found to be affected by this interesting cellular hypertrophy. However, it should be noted that this disease is by no means limited to marine fishes. It has been reported from fresh-water teleosts by other investigators and in the present report is listed as the cause of death of a red-spotted sunfish, *Lepomis humilis* (see Table II).

Among the writer's collection of diseased fishes are several specimens of striped sleepers (*Dormitator maculatus*) (Fig. 14) discovered by Dr. Myron Gordon on one of his trips into Mexico, and a number of pike-perch (*Stizostedion vitreum*) collected by Louis Krumholz from lakes of Illinois. Both species show extensive growths on various regions of the body. Further comparative histo-pathological studies are being made which will be prepared in detail for a later publication.

3. Internal Parasites.

Fishes that die in captivity from internal metazoan parasites usually obtain the infestation in the feral state, since in practically every instance a specific transmitting agent is required. Molluscs and crustaceans are typical intermediate hosts necessary in the life-cycle and transmission of parasites such as digenetic trematodes, tapeworms and spiny-headed worms (Acanthocephala). Because of this fact, parasites belonging to these groups are easily controlled by the simple expedient of removing from the system suspicious intermediate hosts or vectors.

In general it may be said that fishes kept in captivity tend to lose their intestinal parasites after a short time, and these parasites cause little or no damage to the host tissues. Occasionally, however, the infestation may be heavy and the damage produced so extensive that death eventually results. Thus, five striped bass (*Roccus lineatus*) in 1940 and five in 1941 were found to be heavily parasitized with spiny-headed worms (*Echinorhynchus proteus*) (Fig. 3). The worms had perforated the intestinal wall. In some instances, the host tissues reacted to this infestation by walling off the parasite but in other cases the regenerating

tissue was so extensive that an intestinal stenosis developed.

4. Physical and Chemical Factors.

These factors, together with nutrition, account for a large number of deaths among marine fishes and will be discussed again in this paper. Since the nature of these factors is for the most part known, the only criterion necessary to reduce deaths due to them is better management or better equipment. Some factors are beyond control; e. g., sudden increase in concentration of nitrogen gas which appears periodically in certain marine aquaria. The source of this gas is still a mystery. In 1941, gas embolisms were the cause of 61 fatalities of pilotfish, rudderfish, sea bass, croaker, common jack, etc. The condition first appears as extravasation of the surface capillaries, especially in the region of the fins. Exophthalmos is a later manifestation, the result of hemorrhages in the posterior chamber. Just before death, the affected fishes swim in gyrations, turn upside down and finally pass into coma. Examination of brains of these fishes shows cerebral hemorrhages and extensive infarcations (Fig. 4).

5. Violent Deaths.

Fighting, jumping out of the tanks, banging snouts against the wall and glass of the aquaria invariably account for large numbers of mortalities. The interesting phennomenon of leaping demonstrated by tarpons (*Tarpon atlanticus*) resulted in 18 deaths in 1940 and 1941. These fish were sent to the Aquarium by Dr. Breder from Palmetto Key, Florida, when they were several inches long. After surviving the initial shock of handling and transportation, they became acclimatized to the aquarium conditions and in two years measured from 2 to 3½ feet. During this growth period the fish behaved quietly and only occasionally would they break the surface. Suddenly they began to take great leaps, banging their heads and backs on the cross beams supporting the large tanks which contained them. The multiple abrasions received resulted in death.

Schlaifer and Breder (1940) made a detailed study of leaping behavior of young tarpon under various conditions and pointed out that large temperature changes were the important factors involved in this respiratory activity. It is also known that increased density causes an increase in the respiratory rate (see Keys, 1931) in certain marine fishes. Whether this was a factor in inducing tarpon in the Aquarium to leap is not definitely known. At the time that the fish began to leap, the density of the sea-water had reached a specific gravity of 1.030 and when this density was reduced by the addition of fresh-water of same temperature to 1.020 the fish became quiet. It is known that tarpon in fresh water also show the leaping behavior. It may be, as suggested by Dr. Breder, that the two extremes may produce an exhilarating effect, causing the tarpons to jump. Further studies of this behavior in relation to the tonicity of the environment may yield some interesting information.

6. Diet.

In a large and varied collection, the problem of proper feeding is rather difficult. Certain fish require special diets which it is not always possible to obtain. A typical example is the case of seahorses. These interesting fish, with their peculiar mouths, can eat only small food and have a preference for minute crustaceans, such as *Gammarus*, brine shrimp, etc.

The comparatively high incidence of renal and hepatic degeneration is indicative of a faulty diet. There is much comparative evidence to show that such degenerations are involved in vitamin B-complex deficiency. This problem is referred to again in the summary of the factors contributing to major losses of the fishes in captivity.

On the other hand, confinement to relatively small tanks of more or less active fish tends to cause the fishes to lay down enormous amounts of fat. This condition is often found among the older captive fishes, especially those that have been in the tanks from five to ten years. Infiltration and degeneration of fat may occur in the liver, kidney and heart and was probably the cause of death in those specimens so affected.

7. Neoplastic Disease.

Neoplasms of various sorts have been encountered among marine fishes. This item, however, is not as important as a cause of death as it is interesting in the light of similar diseases in humans and other mammals. In fishes, as in human beings, neoplasms are classified according to the tissues or cells involved. The etiological agents responsible for these growths are not always known but they may be due to virus, animal parasites (Neosporidians), heredity, presence or lack of certain chemicals. The nature of some of these fish tumors was reported by the writer (Nigrelli, 1937). Among the tumors, adenocarcinoma of the thyroid appears to be most common among captive fish. In 1940 the writer reported ten cases of thyroid tumor in the sheephead minnow (*Cyprinodon variegatus*). This occurred spontaneously in an established school of fish in one of the tanks but no evidence was obtained to indicate the cause. One interesting case was found in 1940. The fish involved was a blue angelfish (*Angelichthys isabelita*) (Fig. 5) in which the thyroid tumor had grown deep into the

bronchial cavity with an involvement of gill tissues. Histologically, the structure presents a complex network of vascular stroma containing small alveoli without definite lumina and larger, mature alveoli with or without colloid.

B. Summary of Causes of Death Among Marine Fishes.

Table V summarizes in percentage the general causes of mortality among fishes in the Aquarium for the years 1939, 1940 and 1941. It will be seen that there is a sharp decrease from 1939 in the percentage of marine fishes affected by one or more infectious diseases. Concomitant with this decrease, however, was an increase in the percentage of non-infectious diseases, excluding violent deaths, deaths due to abnormal water conditions or unknown factors. Most of the non-infectious diseases occurred in fishes which have been on exhibition for more than one year, and are of the types, as may be seen in Table I, that one normally would expect to find among any animal population of similar proportions and under conditions of confinement.

III. Causes of Death of Temperate Fresh-Water Fishes.

(Table II).

Parasitic diseases took a heavy toll of temperate fresh-water species. This may be partly attributed to certain difficulties encountered with the closed circulation put into use for the first time in the early part of the summer of that year, and the fact that the system's population was suddenly increased by several new collections of cold-water fishes. As shown in Table V the percentage increase of parasitic and infectious diseases was not much greater than in 1939. The rise is due to the fact that physical factors of temperature and aeration were responsible for a significant increase in mortalities for 1940. As may be seen from Table IV the heavy mortalities took place in June and July. Returning the system to an open circulation, especially those tanks containing species requiring lower temperatures and high oxygen content, resulted in an immediate drop in the number of deaths.

The ciliate, *Ichthyophthirius,* as well as the trematode, *Urocleidus* (Gyrodactyloides), and the copepod, *Argulus,* were responsible for 47% of the deaths. These forms had reached epidemic proportions in 1940, and were active during the warmer months when the conditions of crowding and increased temperature were the prime factors in breaking down host resistance. The infestation started on fishes newly acquired in June and the epidemic raged in July with an increased virulency, killing many of the older fishes (average age of 120 fishes, about 4 yrs.). Coincident with the epidemic was the fact that this was a period of abnormal temperatures, both for water and air. The water temperature (July 29) in the closed circulation had reached 77° F. as compared to 67° F. for the water in the open system. Certain fish that had survived the infestation were killed by this increased temperature.

The fishes most affected by the temperature changes were various species of trout, pike and basses. Those most susceptible to *Ichthyophthirius* were the suckers. dace and catfishes (Fig. 7).

As was to be expected, other parasites, which in an open system were insignificant, made their appearance in great numbers when the closed circulation was put into use. Most important among these were the gill and skin flukes (Gyrodactyloides). These parasites were never important, because in a fast flowing, open system, eggs and free-swimming larvae are usually washed to the sewer whereas in a closed system they remained to infect other fishes. There are probably several species of the gyrodactylid forms responsible for the mortalities but insofar as could be determined they seem to belong to the single genus *Urocleidus*. The most susceptible hosts were the members of the Centrarchidae such as the green sunfish (*Apomotis cyanellus*), blue-gilled sunfish (*Helioperca incisor*), warmouth sunfish (*Chaenobryttus gulosus*), rock bass (*Ambloplites rupestris*) and calico bass (*Pomoxis sparoides*).

Fortunately, fish killed by the copepod parasite *Argulus* were limited to the species *Lepisosteus osseus* (long-nosed gars). The bowfins (*Amia calva*) were heavily parasitized by these crustaceans but their resistance was so great that when they were subjected to the salt-water treatment, only one fish died.

In 1941, another parasitic crustacean (*Lernae* sp.) reached epidemic proportions. The infestation had its start on the common goldfish in 1940. In the spring of 1941 other varieties of goldfish were infested and in the early part of the summer the parasites had attacked and killed nine common eels (Fig. 10), and by late summer made their appearance on such fish as brook and common suckers, green sunfish, warmouth, rock bass and small mouth bass. For both types of crustacean parasites, lowering the temperature below their reproductive threshold is an important prophylactic procedure.

A. Other Diseases of Temperate Fresh Water Fishes.

1. Bacterial.

In 1940, a short-lived epidemic of *Bacil-*

TABLE II.

CAUSES OF DEATH OF TEMPERATE FRESH-WATER FISHES.

Parasitic and Infectious Diseases.

DISEASES	1940	1941
Diseases of Skin and Gills		
1. Saprolegnia (Fungus)	37	8
2. Bacillus columaris	14	33
3. Ichthyophthirius (ciliate)	42	1
4. Chilodon (Ciliate)	4	—
5. Cyclochaeta (Ciliate)	6	9
6. Costia (Flagellate)	1	—
7. Ichthyophthirius & Saprolegnia	13	—
8. Ichthyophthirius & Cyclochaeta	10	—
9. Myxosporidia (Cnidosporodia)	5	1
10. Ichthyophthirius and Gyrodactylid	25	—
11. Gyrodactylids (Trematoda)	48	4
12. Argulus (Copepod)	48	1
13. Lernae sp. (Copepod)	9	33
14. Lernae and Gyrodactylid	—	4
Diseases of Skin and Internal Organs		
15. Lymphocystis	1	—
Diseases of Digestive System		
16. Enteritis due to Nematodes	1	—
17. Hepatitis due to Cestodes	—	1
Diseases of Circulatory System		
18. Pericarditis due to Neascus Infection (Trematoda)	1	—
19. Ruptured Sinus venosus (due to Gordiid worms)	—	1
Diseases of Internal Organs (General)		
20. Infectious Lymphosarcoma	8	4
21. Degeneration of Kidney, Liver, and Reproductive Organs due to Strigeid Worms	2	—
22. Degeneration of all Organs due to Gordiid Worms	—	5
Total	275	105

Non-Infectious and Non-Parasitic Diseases.

DISEASES	1940	1941
Neoplastic Diseases		
23. Myxomata	1	—
24. Epithelioma	1	—
25. Osteoma	1	—
26. Ovarian tumor	—	2
Diseases of Digestive System		
27. Prolapsed Intestine with Stenosis	1	—
28. Liver Degeneration	5	1
29. Ruptured Gall Bladder	—	1
Diseases of Urinary System		
30. Kidney Degeneration	7	—
Diseases of Reproductive System		
31. Ovarian Degeneration	8	4
Diseases of Circulatory System		
32. Arterial Degeneration	—	5
33. Internal Hemorrhage	—	1
Diseases of Bone and Organs of Locomotion		
34. Lordosis	1	—
35. Atrophy of Tail Fin	2	—
Diseases Due to Nutrition		
36. Malnutrition	2	—
Senility		
37. Death Due to Old Age	5	—
Death Due to External Causes		
38. Temperature Changes	80	—
39. Causes unknown	41	—
Total Non-infectious Diseases	155	14
Total Infectious Diseases	275	105
Grand total	430	119

lus columaris occurred among the several species of catfishes (Fig. 6), the disease reappearing during the summer months of 1941. The bacilli were first described by Davis (1922) from a variety of fish hosts, including the catfishes. However, Davis had not determined any specific staining reaction for these organisms. In our studies, it was demonstrated that the bacilli are gram-negative in reaction. They are long, slender motile rods and within the measurement range given by Davis (5-12 x.25-.50μ). In the flat-headed catfish (*Ameiurus platycephalus*) and mud catfish (*Opladelus olivaris*), the disease was characterized by multiple circular patches scattered over the entire body, while the lesion in the common bull head (*Ameiurus nebulosus*) was characterized by a single circular growth on the dorsal part of the body, almost in the midline. These growths are grayish in appearance, rimmed by a well-defined hyperemia. Later the skin sloughs, exposing the underlying tissues The lesion never penetrated beyond the skin. An interesting fact concerning this bacterial disease is that once the skin is sloughed, the disease is terminated. However, it reappears on another part of the body unless the fish is removed from the infective tank. Occasionally, a secondary infection with *Saprolegnia* may occur. If the lesions due to *B. columaris* are too extensive, death results. However, in many cases the disease was successfully treated by exposing infected fish to a 1:4000 solution of formalin. The treatment was effective only in the initial stages of the disease. A tank of fifty young bull heads so treated resulted in a complete cure with the loss of only five fish.

2. Saprolegnia.

This fungus attacks fresh-water fishes which have been injured either mechanically or by parasites. Handling thus represents a constant hazard, since spores of this fungus are omnipresent.

3. Lympho-sarcoma.

This neoplastic disease is classified here because of the infectious nature indicated. A number of northern pikes (*Esox estor*) from two to six years old began to die in January, 1940, with other deaths occurring periodically throughout the year. Autopsies revealed large, massive growths on the kidneys and abscesses in the livers. The tumors on the kidneys were, in some instances, grayish and granular in appearance, with practically the entire posterior part of the organ involved. Histologically, this structure presents a diffuse growth of lymphoid cells lying in reticular tissue. There was a certain amount of post-mortem degeneration involved but there could be no doubt that these lesions were lymphosarcomata. In other cases, the growths were nodular (Fig. 9) and also grayish in appearance, which histologically showed an adenoma reproducing renal tubules. The relationship between these two types of neoplasms in the pike, which in some cases were found on the same kidneys, is not known.

The abscesses on the liver (Fig. 8) of diseased pikes were made up of "lymphoid cells" of similar appearance to those found in granulated kidney tumors, indicating that perhaps metastases had occurred. Much of the liver tissue was involved and fish so affected had tremendously enlarged gall bladders filled with a light brown, watery bile. Further studies on this interesting disease are being made and will be published soon.

Piehn (1924) reported the presence of lymphosarcoma in goldfish and adenoma of kidney of pike.

4. Larval Trematodes.

These parasites are the metacercarial stage of a group of digenetic trematodes belonging to the family Strigeidae. The adults of these flukes are found in birds and mammals. Van Cleave and Mueller (1934) have shown that the metacercariae of this family are of three distinctive types recognized as Diplostomulum, Tetracotyle and Neascus. According to these authors, "Each type has a more or less definite location in the host. Thus Diplostomulum is usually . . . found in the eye . . . Tetracotyle is found encysted in the muscles, mesenteries, or pericardium of the host, and is surrounded by a rounded, usually thick, cyst. Neascus has an ovate, thin cyst, and is found on the viscera or mesenteries, in the skin, and frequently in the pericardium." The damage to the tissues produced by the more active worms (Neascus) may be considerable. If a vital organ (liver, kidney) is involved, death will occur. It is of interest to point out that in several cases a heavy infestation was found in the gonads of blue-gill sunfish, producing parasitic castration. Several cases of strigeidiasis (probably Tetracotyle) involving the pericardium of sunfish (Fig. 11) were encountered. It is difficult to say whether pericarditis produced by these encysted worms was fatal even though no other infections or disease were determined at autopsy.

It should be known that these parasites present no problem in aquarium management. Fish obtain the infestation in their native waters and nothing can be done in treating the disease because of the location in the host. Occasionally, as in the case of an infestation of long duration, the host eventually lays down a resistant cyst to wall off the parasites. The cycle is completed only if the infested fish is eaten by a definitive host (bird or mammal).

5. The Gordiacea or "Hairworms."

In 1941 from April to July, the writer reported a number of cases of infestation with hairworms (*Chorodes*). These worms are nemathelminth forms belonging to the Class Nematomorpha. They are larval in character and have been recorded as parasites of insects, annelids, snails and occasionally in man and in fish. Their presence in fish (as in man) must be considered accidental, even though a total of 19 cases were encountered among tropical, semitropical and temperate fresh-water forms. Among the last mentioned group were the brook trout (*Salvelinus fontinalis*), rainbow trout (*Salmo irideus*) and Kentucky bass (*Micropterus punctulatus*). The infestations of the tropical fishes (*Limia dominicensis, Poecilia vivipara, Platypoecilus maculatus* and *Poecilobrycon* sp.) produced greater tissue damage and the fishes had their bellies distended with large amount of serous exudate. In all cases, two worms, sexually immature male and female, were found in the body cavity strongly coiled around each other. The organs of the hosts were greatly displaced and the parasites had set up considerable tissue reactions. In the case of the bass, the sinus venosus was penetrated by the worms, causing hemorrhages.

The source of this infestation was not established. The cycle is not completely understood. The worms become sexually mature while in the free-living state, laying eggs in water which hatch into free-swimming embryos possessing a proboscis and hooks at the anterior end. It is by means of these hooks that the organism bores its way into the body cavity of some aquatic insect (mayfly larva?) or other arthropod. It is not known whether it can bore its way into fish tissue, either through the intestinal tract or through the body wall. In the present instances, aquatic Oligochaeta are the suspected vectors.

6. Cnidosporidian Parasites.

These parasites, as a rule, are not important agents in causing death of aquarium fishes. However, because of the direct mode of infection, they present a continual threat. The five deaths recorded for 1940 were common suckers (*Catostomus commersonii*) and were due to heavy infestations of gill forms belonging to the family Myxosomatidae. The spores were enclosed in cysts found on the gill lamellae. They were oval in shape with two polar capsules and the sporoplasm without iodinophilus vacuoles. Except for the location, the parasites resemble *Myxosoma bibullatum* Kudo (1934) reported from the skin of the same host.

Two cases of infection with the Myxobolidae, *Myxobolus conspicuus* Kudo, were found in the snout of the red horse sucker (*Moxostoma aureolum*). These parasites, however, were not the cause of death of the fish. It is reported here because of the interesting tumor associated with the infection. The disease appeared as a large, smooth growth, and stained sections revealed the presence of typical spores and pansporoblasts in various stages of development in the subdermal regions. Associated with this infection was a definite hyperplasia of the prickle cells of the epithelium. Prickle cell tumors are rare, even among human beings, and further studies are being made. Other interesting tumor-producing myxosporidians were reported by Nigrelli and Smith (1938, 1940) (see Fig 1).

7. Non-Infectious Diseases.

The percentage of deaths due to non-infectious diseases was considerably higher in 1939 than either in 1940 or 1941.

Several interesting cases of neoplasms are reported among which is a myxoma of the skin of rock bass (*Ambloplites rupestris*) (Fig. 13) and "lip" tumors on brook and rainbow trouts (*Salvelinus fontinalis* and *Salmo irideus*). The history of the disease in the rock bass is very interesting in that it may throw some light on the origin of the myxoma. Earlier in the year (1940) the fish was heavily infested with *Chilodon*, a protozoan ciliate, and the body covered with typical grayish mucus. The condition resolved itself and later a translucent, whitish patch remained. It has been observed many times that fishes heavily parasitized with external forms exude tremendous amounts of mucus. An examination of this material usually reveals many cellular elements, other than parasites, among which may be found some cells that exhibit amoeboid movements. In stained preparations the mucus contained stellate-shaped cells and eosinophiles, which are typical components of the myxoma under discussion. Among the 1939 mortalities, 13 cases of myxomata were reported.

The "lip" tumor of the brook trout is a hyperplastic growth of epithelial cells and, therefore, a true epithelioma. A similar case was reported in 1940. The "lip" tumor on the rainbow trout was an exostosis of the tip of the lower jaw bone. Both of these types of tumors are traumatic in origin, the result of the fish banging into the wall of the aquaria (see Fig. 17).

Incidences of prolapsed gut in fishes are not rare. Nigrelli and Breder (1935) reported an interesting case in the tropical fish *Mollienisia latipinna*, in which the prolapsed part of the intestine was completely everted and the lumen filled with cords of cells derived from the serosa. A somewhat similar condition was found in a rock bass (*Ambloplites rupestris*) (Fig. 12). This fish was a seven-year-old female and what brought on the eversion is not known. The bass was healthy in all other respects and was kept alive for several months, later being sacrificed for study.

The five cases of arterial degeneration reported in 1941 occurred in calico bass (*Pomoxis sparoides*) from three to five years old. Hemorrhages through the gills were noted in some of the specimens and while still in a moribund condition the fish were killed and autopsied. The blood vessels were firm and when handled with forceps were found to be friable. Just what is the cause of this "sclerema" is not known. It should be mentioned that so far, there is no evidence to show that fishes are subjected to the types of arteriosclerosis reported for mammals and other vertebrates.

The liver degenerations reported for fishes may be of several types. Most common disturbance is atrophic cirrhosis. In advanced cases very little of the liver pulp is present. Since no infection has been determined, it is assumed that the condition may be due to a deficiency of nutrilites.

The kidney degeneration shows up as a complete breakdown of the organ. In many cases only small bits of kidney tissue remain, usually in the anterior region of the body cavity. Just how such fish carry on the excretory function is not known. There is some evidence in the literature that a certain amount of excretion may take place through the gills. As in the cases of hepatic degeneration, renal damage may be due to dietary deficiency and evidence from experimentation on other vertebrates indicates that they may be due to a deficiency in choline, one of the factors in the B-complex.

IV. Causes of Death of Fresh-Water Tropical Fishes. (Table III).

The tropical forms include some of the smallest of fishes and it is difficult to perform adequate autopsy. These fish are kept

TABLE III.

CAUSES OF DEATH OF TROPICAL FRESH-WATER FISHES.

Diseases	1940	1941
Parasitic and Infectious Diseases.		
Diseases of Skin and Gills		
1. Saprolegnia (Fungus)	1	—
2. Tuberculosis	—	3
3. Ichthyophthirius (Ciliate)	6	13
4. Gyrodactylids (Trematode)	5	2
5. Ichthyophthirius and Gyrodactylids	28	—
Diseases of Circulatory Organs		
6. Ruptured Sinus venosus (due to Gordiid Worms)	1	—
Diseases of Internal Organs (General)		
7. Mechanical Destruction of Internal Organs by Gordiid Worms	—	14
Total	41	32
Non-Parasitic and Non-Infectious Diseases.		
Diseases of Digestive System		
8. Liver Degeneration	6	—
9. Biliary Cirrhosis	—	2
Diseases of Reproductive Organs		
10. Egg bound	—	1
11. Ovarian Degeneration	—	4
Diseases of Circulatory System		
12. Ruptured Myocardium	1	—
13. Splenomegaly	1	—
14. Gas Embolism	—	1
15. Internal Hemorrhage	—	1
Diseases of Organ of Vision		
16. Blindness	2	—
Diseases of Bone and Organs of Locomotion		
17. Swim Bladder Trouble	2	2
18. Lordosis	2	—
Unknown Causes		
19. Edema (see Fig. 14)	3	1
Diseases Due to Nutrition		
20. Malnutrition	11	4
Senility		
21. Death due to Old Age	20	2
Violent and Accidental Deaths		
22. Fighting	33	14
23. Jumped Tank	6	1
Death Due to External Causes		
24. Temperature changes	2	12
25. Changes in Water Chemistry	33	5
26. Unknown	73	38
Total Non-Infectious Diseases	195	89
Total Infectious Diseases	41	32
Grand total	236	121

in tanks with temperatures ranging from 70-80°F. and unless the fish is taken while still in a moribund condition or sacrificed when the disease first appears, it is difficult to make a proper diagnosis since post-mortem degeneration will often cloud the primary cause. It is for this reason that the highest percentage of mortalities are included in the category of causes unknown. This is shown in Table V. However, it will be noted that there was a great increase in infectious diseases for both 1940 and 1941. The agents for these deaths were limited as to kind, being typical of the forms one would expect to find among any fish collection. The ciliate, *Ichthyophthirius*, and the monogenetic fluke of the group Gyrodactyloides, are responsible for the greatest number of mortalities.

In 1940, three cases of tuberculosis were found in platyfish, *Platypoecilus maculatus*. The disease was known to occur in this fish for some time but it was only recently described in detail by Baker and Hagan (1942). The organism has been identified as a specific agent, *Mycobacterium platypoecilus* and differs from all other strains of known acid-fast bacteria, except one which lives as a saprophyte in the soil. These investigators were able to culture the organisms on glycerol phosphate agar at room temperature and at 37°C. They also were able to reproduce the disease in normal platys and goldfish with the cultured material.

In 1942, an epidemic of tuberculosis broke out among other tropical fishes which included the following forms: neon tetra (*Hyphessobrycon innesi*), white cloud mountain fish (*Tanichthys albunuches*), pearl danio (*Brachydanio albolineatus*), spotted danio (*Brachydanio analipunctatus*), and zebra danio (*Brachydanio rerio*). The lesions found on these fishes were typical, externally appearing as whitish patches on the dorsal side of the body. The growths were first seen on the head and gradually spread over the entire skin. Stained smears from skin lesions demonstrated large encapsulated rods usually found in cells identified as macrophages (Fig. 15). However, the organisms varied considerably in form and size, and in staining reaction. Some were minute, almost round in shape, while others were long and slender. The longer rods were only faintly colored with acid fuchsin. Histological examination of all the internal organs indicated the presence of these pathogens, particularly in the kidney, spleen and liver. The lesions produced were more pronounced in the kidneys, where much of the tissue was involved.

Whether the acid fast organisms found in these fish are the same as those found in the platy was not determined. Attemps to culture them have so far been unsuccessful but further studies are being conducted to determine this point and also their origin.

As was mentioned previously, water conditioning is a prime factor in maintaining

these small, fresh-water tropical fishes. In both 1940 and 1941, 14% of the deaths were attributed to conditioning disturbances.

V. Summary of the Factors Contributing to Major Losses of Fishes in Captivity.

As may be seen from Tables I-III, the causes of diseases and death of fishes in captivity are numerous and by no means limited to infections by animal or by plant parasites. Epidemiological studies among aquatic vertebrates have revealed that the factors involved are about the same as one would expect for human populations or for any other group of animals. The following are some of the more important physical, chemical and other factors to consider in preventing major catastrophies of fishes kept in captvity.

1. Crowding.

Breder and Coates (1931) have established the fact that there is a definite population density of fishes for a given volume of water. They showed that when the ratio of volume of fish to volume of water (surface area remaining constant) exceeds the optimum density, fish will kill each other off to maintain the population at equilibrium, when all other ecological factors are equal.

Any increase in numbers of fish (total volume of fish remaining the same) offers more body surface which may be attacked by parasitic agents. Ordinarily, there may be a few parasites present on the body, which under conditions of crowding will increase in numbers to the extent that the minor effects of the individual parasite is multiplied many times and the vitality sapped exceeds the margin of safety. As an immediate result of crowding, the mortality of the host species increases greatly and the epidemic rages until the contaminated tanks are depleted or an immunity is developed by the host.

2. Temperature.

Fishes, particularly those of the temperate fresh-water variety, can withstand a comparatively wide range of temperature, but only if the changes are gradual. For certain psychrophilic and thermophilic types, the temperature range tolerated, even though the change may be gradual, is much restricted. In all groups of fishes, sudden change of temperature, even of a temporary nature, is a great shock to the fish and often fatal.

Fishes subjected to sudden temperature changes, small or large, very often become susceptible to infestation by one or more of the parasitic ciliates (*Ichthyophthirius*, *Chilodon*, Trichodina, etc.). The rapidity with which these parasites appear on the skin of fishes after such a change is as yet not thoroughly understood. In the case of *Ichthyophthirius*, the writer believes that a drop in temperature induces encystment. It is in the encysted stage that the ciliate undergoes multiplication, each individual cyst capable of producing hundreds of infective organisms. MacClennan (1937) showed that the transformation of ciliophores which are unable to encyst or divide to mature stages which can encyst and produce infective individuals is a very rapid one. Increasing the temperature induces excystment, resulting in the liberation of the young ciliophores. These young forms are very susceptible to changes in osmotic pressure so that adding salt-water to make up a solution of one-half strength (sp. gr. about 1.0150), or sodium chloride to make up a solution of .5%, will kill these organisms (and other ectoparasitic protozoa or flukes) with little or no effects to the host.

Since it is known that reproduction among organisms is definitely correlated with temperature, the ability to control this factor at any definite point becomes an important prophylactic measure in aquarium management.

3. Light.

That an optimum amount of light is an essential requirement to maintain fishes in good health is quite evident. Breder and Harris (1935) have analyzed the effect of light on orientation and stability of certain fish species, particularly young filefish and young cowfish. If such fish are exposed to a beam of light, they lose all sense of orientaion and move about in rapid gyrations. As to the harmful effects of strong light, they state, "In fact it seems to be definitely established that violent or continuous changes in orientation of fish is decidedly harmful, if not indeed fatal to the fish."

The relationship of light disease-producing agents is not definitely known. There is much evidence in the literature concerning the phototropic reaction of parasites. Increasing the amount of available light over the fresh-water tanks was followed by an epidemic of lernaiasis and may be partly attributed to this factor, especially in the movements of the larvae of these fixed parasites. It is a known fact that larval trematodes are positive-phototropic and may account for the predominance of monogenetic forms on surface fishes.

4. H-ion Concentration.

The range of the degree of acidity or alkalinity (pH) that fishes can tolerate varies considerably with the species. The optimum pH value for marine forms is around 8, while the optimum for fresh-water species is around the neutral point

(pH 7.0). As will be shown, fishes can and do control, to a certain degree, the chemical contents of their environment .That they play some part in adjusting the pH of their environment can be shown by taking a pH reading of water before and after fishes have been included. Comparatively few species of fishes are favored by completely acid water (i.e., below pH 7) ; most of them live in water on the alkaline side of the pH range.

The chemical control of sea water for aquaria has been thoroughly established by Breder and Howley (1931) and the technique perfected by these investigators is now in use in most aquaria. They point out the necessity of a careful check on the bicarbonate salts. These are buffering agents needed to maintain the reaction at a proper level but which are continually broken down by the acid metabolites of aquatic organisms.

5. Specific Gravity.

The optimum density of sea-water for the proper maintenance of marine animals is established by the density under natural conditions. The harmful effects on the tissues of fishes of changes in density and salinity of water has been studied by Sumner (1906) and more recently by Keys (1931). The latter worker has shown that an increase in density raises the respiratory rate, hence increases metabolic activity. Many species can tolerate abrupt changes in density, and while prolonged exposure to high or low densities do produce deleterious effects, the degree of damage may vary considerably with the species. This is accounted for, in part, by the fact that in the former case the body tissues have had no time to make adjustments for the differences in osmotic pressure, and nothing is lost or taken in. Invertebrates, however, cannot tolerate even comparatively slight changes in density and salinity and for this reason are extremely difficult to keep under aquarium conditions for any length of time.

Since fishes have a greater tolerance than parasites (invertebrates) for changes in the tonicity of their environments, the control of the specific gravity also becomes an important prophylactic and therapeutic measure.

6. Flow and Aeration of Water.

Some fish require fast moving bodies of water with a high oxygen content. Any depletion in the oxygen threshold for fishes of high metabolic activity may be fatal. Methods of controlling the oxygen supply or the reduction of tension of other and more harmful gases are many. Artificial areation, green plants, increasing the surface area, increasing the flow, lowering the temperature, etc., are all aides in keeping the oxygen tension at optimum level. Too much oxygen may be just as detrimental as too little of this important gas. It is a known fact that fishes may develop gas embolisms, producing hemorrhages in various parts of the body, especially of the surface capillaries. Chlorine and nitrogen are other gases which may be present in water and which can be toxic in certain concentrations.

7. Metabolic Waste Products.

It is known among Ecologists that fishes living in small standing bodies of water alter the chemical content of their environment, apparently not only making it more suitable for themselves, but sometimes rendering it completely toxic for even closely related species. This has been called water conditioning. It is a very important factor in maintenance of fish in captivity, especially the small fresh-water tropical varieties. Just what this conditioning is, has so far evaded successful analysis. Evidence indicates that some specific animal protein or protein derivatives may be involved. Among marine species it is known that a tank of elasmobranchs will produce a sufficient concentration of metabolic waste materials to be toxic for many teleost species when placed in it.

8. Diet

Many diseases of fishes kept in captivity are due to vitamin deficiencies. Raw fish, both frozen and fresh, forms an important item in the diet of fishes kept in captivity. Fishes placed entirely on such a diet will show certain definite symptoms, such as loss of appetite, excitability, loss of coordinated movements, etc. According to Wolf (1942), similar symptoms developed in trout under experimental conditions were alleviated by the addition of vitamin B_1 to the diet.

As was mentioned above, the large number of liver and renal damages encountered during autopsies may lead to an inference of a deficiency of certain other essential nutrilites. Since it has been determined that choline is indicated for similar conditions in higher vertebrates, experiments are now under way to determine the efficacy of this compound for these diseases in fish.

9. Handling and Permanency of Location.

Handling is a very important item and directly or indirectly may be responsible for the deaths of large numbers of fishes. Fish handled will invariably receive abrasions which become the focal point of infection with bacteria or molds. If the injured surface area is large, death will result.

Fishes collected during the spawning season are a poor risk as exhibition speci-

TABLE IV.

COMPARISON BY MONTH OF THE NUMBER OF DEATHS OF FISHES FOR 1939, 1940 AND 1941 (8 MONTHS ONLY).

	1939			1940			1941		
	M	T	Tr*	M	T	Tr	M	T	Tr
Jan.	25	0	21	34	18	19	33	10	10
Feb.	43	6	15	34	27	23	24	7	18
Mar.	40	0	34	20	16	13	26	6	18
Apr.	47	2	24	30	33	36	26	13	21
May	67	14	43	84	7	19	29	6	10
June	78	7	16	63	120	14	27	13	9
July	328	34	45	63	126	22	17	43	17
Aug.	115	7	18	46	26	31	13	21	18
Sept.	62	6	21	35	18	16			
Oct.	52	9	12	35	16	23			
Nov.	38	54	15	23	4	13			
Dec.	32	71	12	26	19	8			
Total	927	210	276	493	430	236	195	119	121

* M, Marine; T, Temperate Fresh-water; Tr, Tropical Fresh-water.

TABLE V.

SUMMARY OF MORTALITY PERCENTAGE BY CAUSES.

CAUSES	1939	1940	1941	1939	1940	1941	1939	1940	1941
Infectious	68.00	40.80	37.00	60.50	64.00	88.20	13.90	17.00	26.50
Non-Infectious	13.00	20.50	25 00	20.50	7.90	11.80	31.10	21.00	15.00
Violent Deaths	4.00	7.90	18.00	.50	00.00	00.00	1.10	17.00	12.40
Water Conditions*	11.00	17.20	2.00	13.30	18.60	00.00	31.10	14 00	14.10
Unknown	4.00	13.60	18.00	5.20	9.50	00.00	32.80	31.00	32.00

* Includes density, Temp., pH and Chemical Composition.

mens. They do not successfully withstand rough handling and long truck or boat trips. Many fresh-water species spawn in the spring, on the rise in temperature. Such fish have not recovered from the winter's starvation, and are usually heavily parasitized. Fish that spawn in the fall, i. e., on the drop in temperature, are a better risk, for they have had an opportunity to build themselves up and therefore can withstand the rigors of capture more successfully. Because of the temperature factor, cold water forms should not be collected during the summer months unless proper refrigeration facilities are available.

Once a fish or group of fishes become established in a tank, removing them to other sites results in a high incidence of deaths. In spite of the fact that the water is the same (homotypic) in their new tanks, the fishes become highly nervous, dash about and bang their snouts against the walls. They finally settle to the bottom, refuse all food and eventually die.

Another interesting reaction to permancy of location is the behavior of long-established inmates of a particular tank to new members introduced, even of the same species. The new fish, regardless of size, are often attacked and severly injured or even killed by the established fish or fishes.

10. Parasitism.

All fishes should be examined for parasites, especially external forms. The importance of this procedure is obvious and has been reported by Nigrelli and Atz (1943). It should be pointed out further that under epidemic conditions, many of the external parasites show no host specificity and the virulency is increased many fold.

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EXPLANATION OF THE PLATES.

(All pictures by S. C. Dunton, Photographer N. Y. Zool. Soc.)

Some Diseases of Marine Fishes.

Plate I.

Fig. 1. *Cyprinodon variegatus* with myxosporidian (*Myxobolus lintoni*) tumors. 3 ×.

Fig. 2. *Epibdella melleni*. Round pompano (*Trachinotus falcatus*) heavily parasitized with this monogenetic trematode. About 2 ×.

Plate II.

Fig. 3. *Echinorhynchus proteus* (Acanthocephala). Intestine of striped bass (*Roccus lineatus*) showing a heavy infestation with these spiny-headed worms. Note perforations of the intestinal wall and thickened mucosa. About 2 ×.

Fig. 4. Gas embolism. Brain of common jack (*Caranx hippos*) with hemorrhagic infarct. About 2 ×.

Fig. 5. Thyroid adenoma. Head of blue angelfish (*Angelichthys isabelita*) with thyroid tumor invading the gills. About ½ natural size.

Some Diseases of temperate Fresh-Water Fishes.

Plate III.

Fig. 6. *Bacillus columaris*. Mud catfish (*Opladelus olivaris*) showing typical dermatitis due to this bacillus. About ⅓ natural size.

Fig. 7. *Ichthyophthirius multifilis*. Channel catfish (*Ictalurus furcatus*) showing typical pustules on the skin. These are the encysted ciliates (Holotricha) which in time will burst open, each cyst releasing about 200 minute ciliophores. About 3 ×.

Plate IV.

Fig. 8. Infectious lympho-sarcoma. These liver abcesses in the pike (*Esox estor*) are associated with large granular masses involving the greater bulk of the kidney. Histologically, the cellular elements in both of these growths are mainly round cells. The infectious nature is indicated by the fact that several pikes of different ages autopsied at about the same time were found affected by this disease. Note enlarged gall bladder. About ⅔ natural size.

Fig. 9. Kidney adenoma. Large nodular tumor in kidney of pike which histologically showed reproducing renal tubules. About ⅔ natural size.

Fig. 10. *Lernae* sp. Head of eel (*Anguilla rostrata*) split open to show the heavy infestation of this crustacean parasite (Copepoda). The parasites were mainly localized in the mouth cavity. About natural size.

Fig. 11. Strigeid flukes. Larval trematodes (Tetracotyle) encysted in the pericardium of common sunfish (*Lepomis gibbosus*). About 3 ×.

Plate V.

Fig. 12. Prolapsed gut. The protruding intestine shown in this rock bass (*Ambloplites rupestris*) is completely occluded by cellular elements derived from the serosa. About ½ natural size.

Fig. 13. Myxoma. A mucoid growth on a rock bass which histologically shows the presence of typical cellular elements (stellate shaped cells and eosinophiles) usually found in these tumors. About ½ natural size.

Some Diseases of Tropical Fresh-Water Fishes.

Fig. 14. Lymphocystis. This disease is characterized by a hypertrophy (increase in size) of connective tissue cells. The nodular growths shown in this striped sleeper (*Dormitator maculatus*) are made up of groups of these giant cells. 2 ×.

Plate VI.

Fig. 15. *Mycobacterium* sp. (*M. platypoecilus?*) from skin of Neon tetra. Note bacilli in large macrophage cells. 1800 ×.

Fig. 16. Edema. This disease is of common occurrence in many of the smaller tropical fresh-water fishes. The exact nature or cause of the disease is not known. In some, it is characterized by the presence of large amounts of serous exudate in the body cavity. In others, for example, the blind cave characin (*Anoptichthys jordani*), the material found in the body cavity is a translucent gelatinous-like substance of unknown nature. About natural size.

Fig. 17. Lip tumor. Many fish in captivity develop lip tumors as a result of banging continuously into the walls of the tank. These tumors, like the one shown here in *Acestrorhynchus microlepis*, may be either osteomata or epitheliomata in character. About 3 ×.